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Milk Fever *

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YOU may be somewhat surprised that one whose main official function is to instruct in the maintenance of health and the prevention of disease has chosen as his subject this afternoon an entity which is strikingly removed from the state of health and about whose prevention we still know little or nothing. However, those of you who remember the "Dick" a quarter of a century ago, when Dryerre and Greig had just launched their parathyroid deficiency theory on the stormy seas of controversy with the subsequent disputes about the relative merits of inflation and injection, will not be surprised that a very junior student of those days has retained a vivid interest in the subject ever since. Moreover, a few years ago also, I was injudicious enough to make a few observations on the subject at a Divisional meeting in Edinburgh (Robertson, 1949), which resulted in some of my younger colleagues being stimulated to undertake further investigations on the subject. That work is partly published and partly in the press so I felt it would not come amiss to tell you briefly what we have done and to take another look at the problem in the light of our findings to date. I ought, perhaps, to warn you at this stage that we have made no revolutionary discoveries and that you will not leave this hall with any panacea, either preventive or curative. But I do hope that what I am going to tell you of our findings in the laboratory will stimulate some of you to tell me of yours in the field; for it is only by co-operation between the laboratory and the practitioner that we can hope to make real progress in the investigation of this or any other disease—and nothing but good can come from getting together to share and discuss our experiences.

* Based on an address given by Professor Robertson to the North of Ireland Veterinary Association on February 4th, 1955.

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Historical

Though milk fever has been known for at least 200 years, and though about 30 different theories have been advanced to account for its occurrence, there are only two landmarks of which I need remind you at this stage. The first occurred in 1897 when Schmidt of Kolding in Denmark, through an erroneous observation married to a false deduction, produced a cure for milk fever in the form of the intramammary injection of a weak K.I. solution which reduced the mortality rate from 60 to 70 per cent. to about 15 per cent., later to be reduced still further by udder inflation. Some 30 years later came the hypocalcaemia work of Little & Wright (1925), Dryerre & Greig (1928), and later of numerous others which was to place the therapy of this disease on an apparently more rational basis, by substituting calcium injections for udder inflation and thus eliminating the hazards of the latter procedure. Once this was widely accepted as a specific method of achieving a rapid, safe and complete cure, it doubtless seemed to many that the milk fever problem was effectively disposed of, though the scientists might still wish to dabble in the whys and wherefore in their never-ending search for ultimate causes. However, the increasing occurrence of cases which did not respond typically to calcium therapy, and the occasional report that udder inflation had proved successful where calcium therapy had failed, decided us to have another look at the problem from both its clinical and aetiological aspects. Preliminary observations I reported a few years ago in THE VETERINARY RECORD (Robertson *et al.*, 1948) suggested that hypocalcaemia was not the whole story; and though they did not bear out current theories about the rôle of blood magnesium in symptomatology, they did suggest that the inorganic phosphate fraction warranted further attention and stimulated the investigations I am going to describe.

Blood Changes at Parturition

Our first investigation was made on the changes in blood calcium, magnesium and inorganic phosphate in normal cows at parturition, partly to clear up some of the contradictions in the literature and partly in the hope of getting a better understanding of the rôle of these substances in milk fever itself. This was done in 32 normal cows on two farms, sampled daily from about a fortnight before to about a fortnight after calving with much more frequent sampling for a few days immediately following parturition.

As recorded in detail elsewhere (Moodie *et al.*, 1955), we were able not only to confirm the observations of previous workers on these changes but to show that they were significantly affected by the number of previous calvings (Table I). The lowest

TABLE I

BLOOD CHANGES AT NORMAL PARTURITION
Percentage Change from Two Days Prepartum

Group	Number of animals	Serum Ca. percentage fall	Blood In. phosphate percentage fall	Serum Mg. percentage rise
First calvers	7	11·7	33·8	7·7
Second calvers	8	17·6	38·4	24·1
Third calvers	9	22·9	46·4	34·4
Fourth and subsequent calving	8	30·6	48·1	35·9

values recorded in these normal animals were 5.65 mg. per cent. for calcium and 1.55 mg. per cent. for inorganic phosphate, while the magnesium rose as high as 4.65 mg. per cent. These figures are, interestingly enough, within the range of values recorded in clinical cases of milk fever.

In general, the results of this investigation, therefore, confirmed the picture observed by previous workers on the behaviour of calcium, inorganic phosphate and magnesium at parturition and suggested that those who differed had probably been taking their blood samples too infrequently. The most interesting part, however, was the finding that the changes in all those constituents become significantly greater with each succeeding parturition. In the light of the known age distribution of milk fever and its usual time of occurrence we felt, therefore, that we were warranted in regarding milk fever to all intents and purposes as an exaggeration of these normal physiological changes occurring at parturition in perfectly healthy ruminants. Interestingly enough, and in keeping with the above concept, we got two borderline cases (Fig. 1) where the animals appeared dull and uneasy after calving and looked as if they were going to develop milk fever, but they gradually returned to normal without developing characteristic clinical signs of the disease. The blood picture there showed that the various ions studied had been about the lower limits of our normal range.

The acceptance of the theory that milk fever is a pathological exaggeration of a normal physiological condition does not take one very far, however, apart from suggesting that an understanding of the causes of the normal changes might help us to understand the milk fever picture. So we next turned our attention to possible causes, starting with the fall in blood calcium, the simplest and probably the most generally accepted explanation of which is that it is due to removal of calcium from the blood by secretion of colostrum. As this, however, had by no means been proved, we thought it might be worth while studying the effect of the onset of lactation on the blood levels by interfering with it—in the one case by starting milking before the normal time, and in the other by stopping milk secretion altogether.

Prepartum Milking

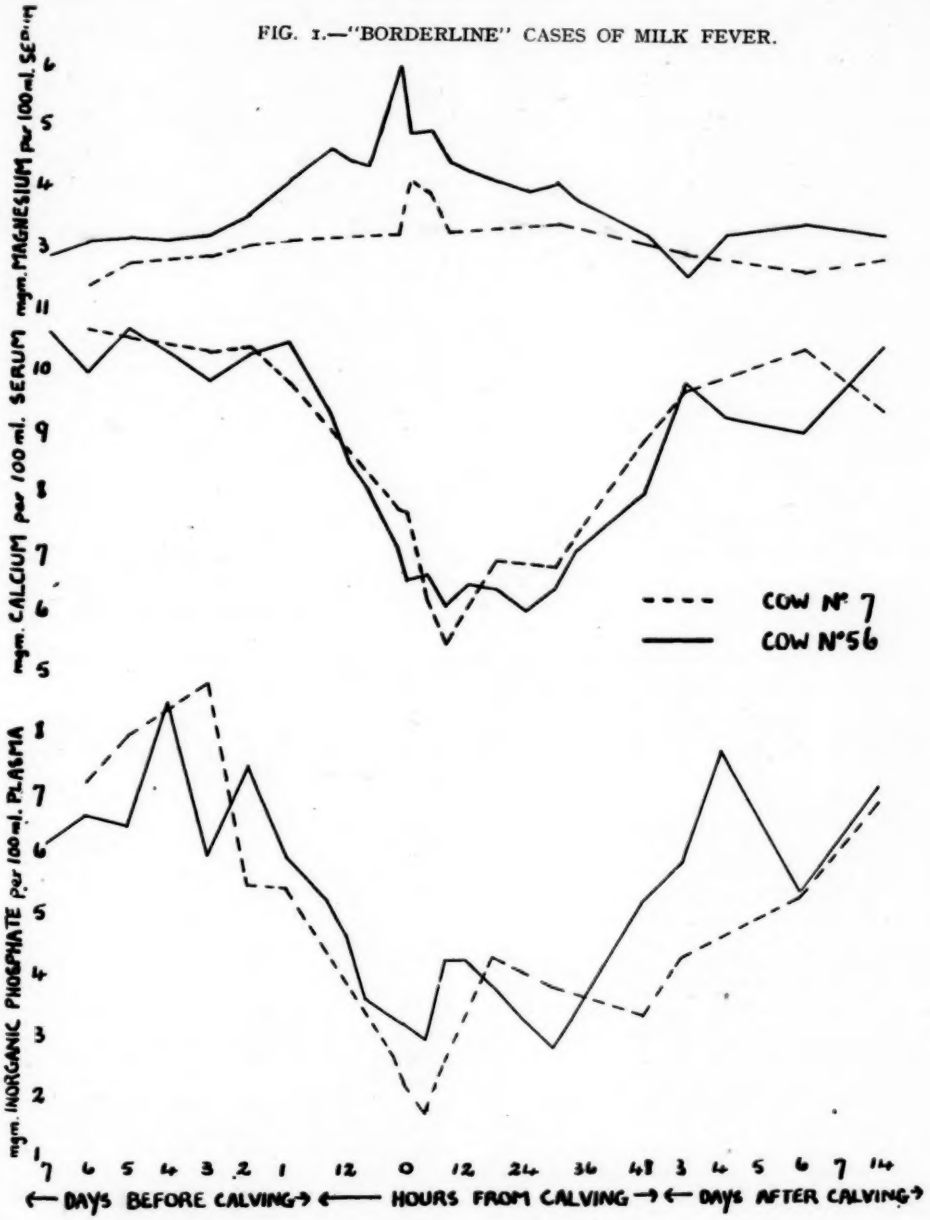
Our prepartum milking experiments were not as satisfactory as we might have wished. We used seven cows and started milking about three weeks before the expected calving date, but in spite of liberal " steaming up " and frequent stripping of the udder, only two gave appreciable quantities of milk before calving (16 gallons and 10 gallons respectively). The others behaved more or less as normal cows in so far as their blood changes were concerned, save that two developed milk fever. Now the results on two animals which milked well prepartum do not warrant rigid conclusions but they were consistent and may be of interest, as most of the published work on this aspect has involved too infrequent blood sampling to be of real value.

Fig. 2 shows the mean data for serum calcium for the two prepartum milked cows compared with that from eight normal animals of the same calving group. You will notice a gradual fall during the last 8 to 10 days prepartum coincident with the rise in the level of milk calcium secretion, and the fall was, if anything, greater than normal. A similar difference was shown by the inorganic phosphate (Fig. 3), in which case you will notice also that the recovery phase was markedly delayed compared with normal cows. Again with serum magnesium (Fig. 4) the rise in the prepartum milked cows tended, if anything, to commence earlier and to be somewhat more sustained than in normal cows of the same age. So far as they go, therefore, these results suggest that the artificial initiation of lactation before parturition does have an effect on the levels of these blood constituents; and that the fall in the Ca. and In. Phosphate fractions is coincident with and more or less proportional to the increase in secretion of these elements in the milk, suggesting that the blood is being depleted by the demands of lactation.

Mastectomy

As a further check on this hypothesis three normal pregnant cows had their udders removed in the hope of differentiating between the changes associated with the act of parturition *per se* and those associated with the onset of milk secretion. The results of blood analysis on these animals compared with the mean data for three normal cows comparable in number of calvings is shown in Fig. 5. You will note only a slight drop in serum calcium at parturition with a rapid return to normal and there was little or no change in serum magnesium. The behaviour of

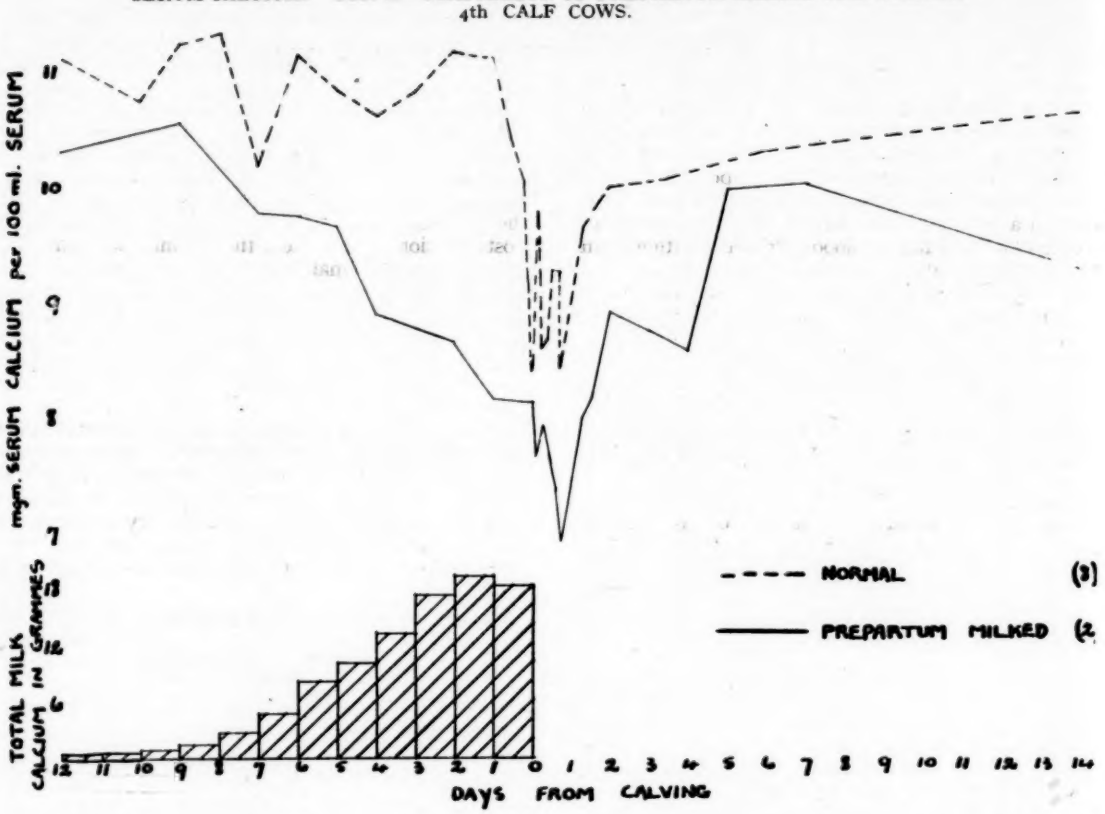
FIG. 1.—"BORDERLINE" CASES OF MILK FEVER.


mgm. MAGNESIUM per 100 ml. SERUM
mgm. CALCIUM per 100 ml. SERUM
mgm. INORGANIC PHOSPHATE per 100 ml. PLASMA
COW N° 7
COW N°56
7 6 5 4 3 2 1 12 0 12 24 36 48 3 4 5 6 7 14
← DAYS BEFORE CALVING → ← HOURS FROM CALVING → ← DAYS AFTER CALVING →

plasma phosphate, however, in the two groups was more or less similar. American workers (Hibbs *et al.*, 1951) have reported similar results for this type of study, which I think we can take as strongly supporting the view that the onset of milk secretion has a very definite influence on the level of blood calcium. In other words, our results suggest that the drain of calcium into the colostrum is a major factor in producing the lowered blood calcium in normal cows at parturition.

The behaviour of serum magnesium would seem to be closely linked with that of calcium, though quite frankly we do not know enough yet about the mechanisms which control the level of blood magnesium. We do know, however, that if you lower the blood calcium experimentally, *e.g.* by oxalate poisoning, you get a rise in blood magnesium; and if you induce an experimental hypermagnesaemia you get a concomitant hypocalcaemia. Our general findings tend to fit in with such a reciprocal relationship and suggest

SERUM CALCIUM. FIG. 2.—COMPARISON OF PREPARTUM MILKED AND NORMAL 4th CALF COWS.



that it may be of a more or less quantitative nature.

We have not, however, been able to demonstrate any such close interrelationship between the blood calcium and the inorganic phosphate. The different behaviour of the two at normal parturition, which we have referred to elsewhere (Moodie *et al.*) with the sudden marked return to normal of the In. Phosphate and later secondary fall, and the results in mastectomised animals both tend to suggest that variations in the inorganic phosphate fraction are more intimately linked with the act of parturition *per se* than are the changes in serum calcium; though the changes in our prepartum milked cows as well as in mastectomised animals (which showed a higher blood phosphate after calving than did the normal controls) both suggest that milk secretion does have some effect on this fraction also, it would seem that the effect of parturition is probably the more important factor in producing the drop in inorganic phosphate at the time of calving.

Now if these changes I have described occur in both normal and abnormal cows, why do some get milk fever and others not? We must, of course, keep in mind the possibility that a lowered level of blood calcium, either by itself or along with the concurrent fall in the inorganic phosphate and rise in serum magnesium, may not be the only requisite for the production of milk fever but that other changes must occur at the same time. But no satisfactory evidence has yet been produced in support of any other suggested factor so I do not propose to speculate further along these lines. Focusing attention on the hypocalcaemia, therefore, we have two possibilities to keep in mind:—

(a) That the fall is greater in milk fever cows than in normal cows of the same age distribution;

(b) That there may be considerable individual variation between cows in the drop of serum calcium which can be tolerated without the exhibition of clinical signs.

The second point I have already hinted at earlier when I mentioned that some of our normal cows at parturition had blood calcium levels within the range commonly taken as causing milk fever, *viz.*, below 7 mg. per cent. and as low as 5.65 in one case.

On the other hand, taken by and large the average fall in blood calcium in milk fever is greater than the mean figures for the maximum decrease in our normal cows. Thus Greig (1930) reported in 82 cases of milk fever minimum, maximum and average serum calcium values of 3.0, 7.76 and 5.13 mg. respectively per 100 ml. and a survey we have made showed similar results, *viz.*, 2.43, 8.20 and 5.01 mg. per 100 ml. But there is no evidence that cows which take milk fever secrete more colostrum than those which do not, nor is there any suggestion that their

colostrum contains more calcium. In other words, there is no reason to postulate a greater loss of calcium by drainage in such animals, so we must conclude either that they are less well able to mobilise their reserves or that they have less reserves to mobilise.

To take the former idea and apply it to our results would suggest that the mechanisms controlling the mobilisation of calcium become less effective with each successive parturition; and that a point is reached usually after the third and subsequent parturition when in a proportion of animals the mechanisms fail to counteract the fall in blood calcium resulting from the onset of lactation to such an extent that clinical milk fever ensues. To go further and ask what these mechanisms are, however, takes us no further in the current state of physiological knowledge than the 30-year-old theory of parathyroid deficiency of Dryerre & Greig (1925) which still lacks experimental support, while attempts to treat milk fever cases with parathyroid hormone have not been encouraging.

The other possibility, that the reserves may not be adequate in some animals and get less adequate as the animals grow older rather suggests the dietetic aspect which has been the subject of considerable controversy and conflicting results. Heavy feeding, steaming up, diets high in phosphorus, and/or deficient in calcium, lack of vitamin D and abnormal Ca : P ratios have all been cited at one time or another as the cause of milk fever. The verdict in all of them must, I think, be " not proven " and it is difficult to square these or indeed any other theory yet propounded with the fact that equilibrium is apparently restored rapidly following successful treatment.

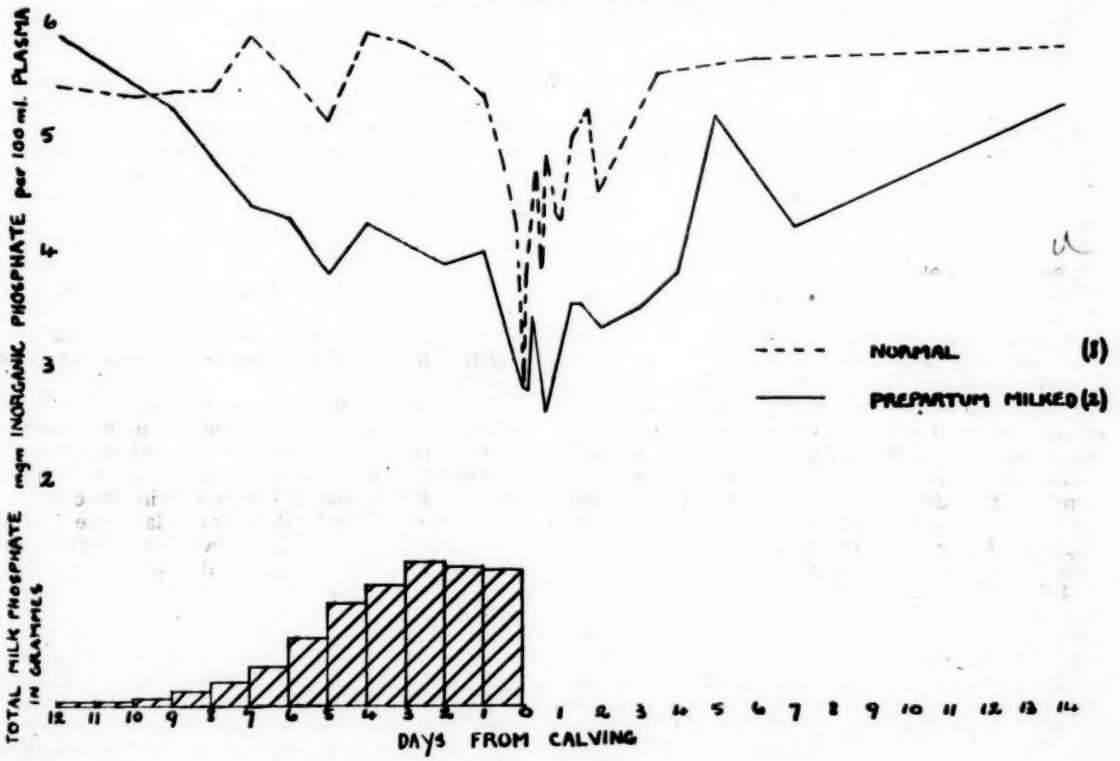
Now here are two individual cases which show some points of interest.

Fig. 6 (Case 61) shows a typical satisfactory response to calcium therapy. The animal was given 2½ oz. calcium borogluconate intravenously and though the calcium fell rapidly from its immediate post-injection level to less than 5 mg. per cent. the cow remained normal so that recovery was uneventful. You will note also a prompt rise in inorganic phosphate with a secondary fall and then a slow but steady rise thereafter more or less parallel with the rise in serum calcium. The blood magnesium also showed a slight tendency to rise in this particular case.

Fig. 7 (Milk Fever Case No. 44) on the other hand shows the type of fluctuation you are liable to get in animals which do not respond normally. Here two intravenous injections of calcium with udder inflation in between failed to give a satisfactory result but a second inflation of the udder more than two days after the first treatment finally got the animal back to normal.

In these two cases one cannot help remarking on the apparently transient effect of intravenous calcium

INORGANIC PHOSPHATE. FIG. 3.—COMPARISON OF PREPARTUM MILKED AND NORMAL 4th CALF COWS.


mgm INORGANIC PHOSPHATE per 100 ml. PLASMA
6
5
4
3
2
NORMAL (1)
PREPARTUM MILKED (2)
TOTAL MILK PHOSPHATE IN GRAMMES
12 11 10 9 8 7 6 5 4 3 2 1 0 1 2 3 4 5 6 7 8 9 10 11 12 13 14
DAYS FROM CALVING

SERUM MAGNESIUM. FIG. 4.—COMPARISON OF PREPARTUM MILKED AND NORMAL 4th CALF COWS.

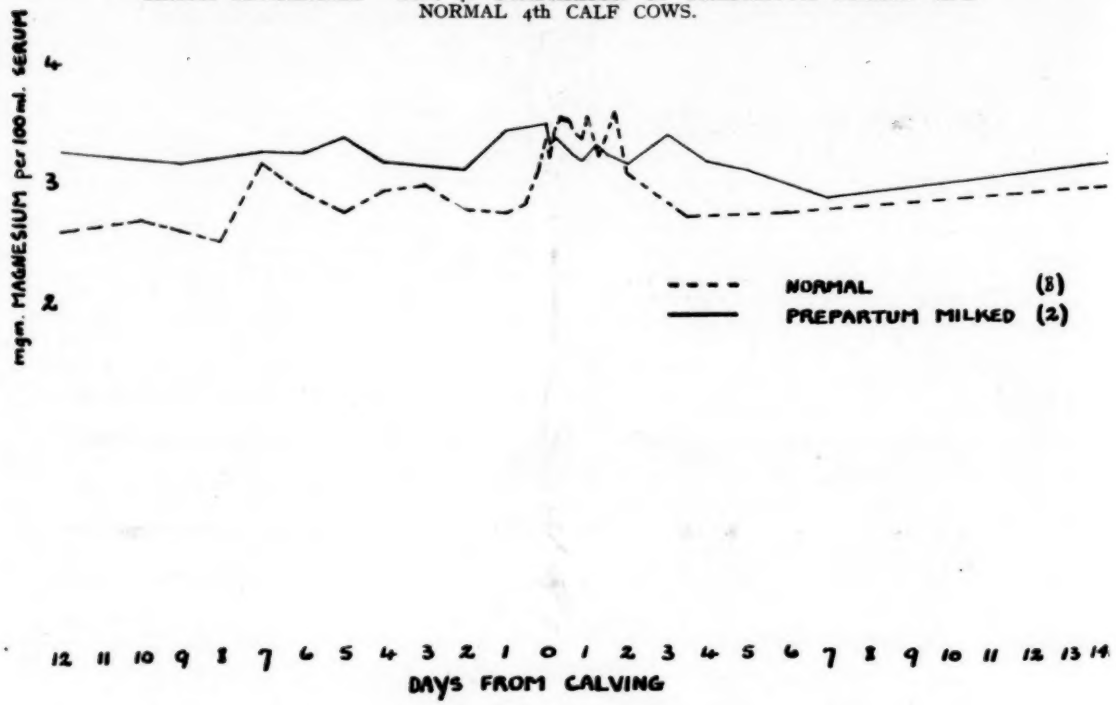

mgm. MAGNESIUM per 100 ml. SERUM
4
3
2
NORMAL (8)
PREPARTUM MILKED (2)
12 11 10 9 8 7 6 5 4 3 2 1 0 1 2 3 4 5 6 7 8 9 10 11 12 13 14
DAYS FROM CALVING

FIG. 5.—MEAN DATA FOR MASTECTOMISED COWS COMPARED WITH MEAN DATA FOR NORMAL COWS.

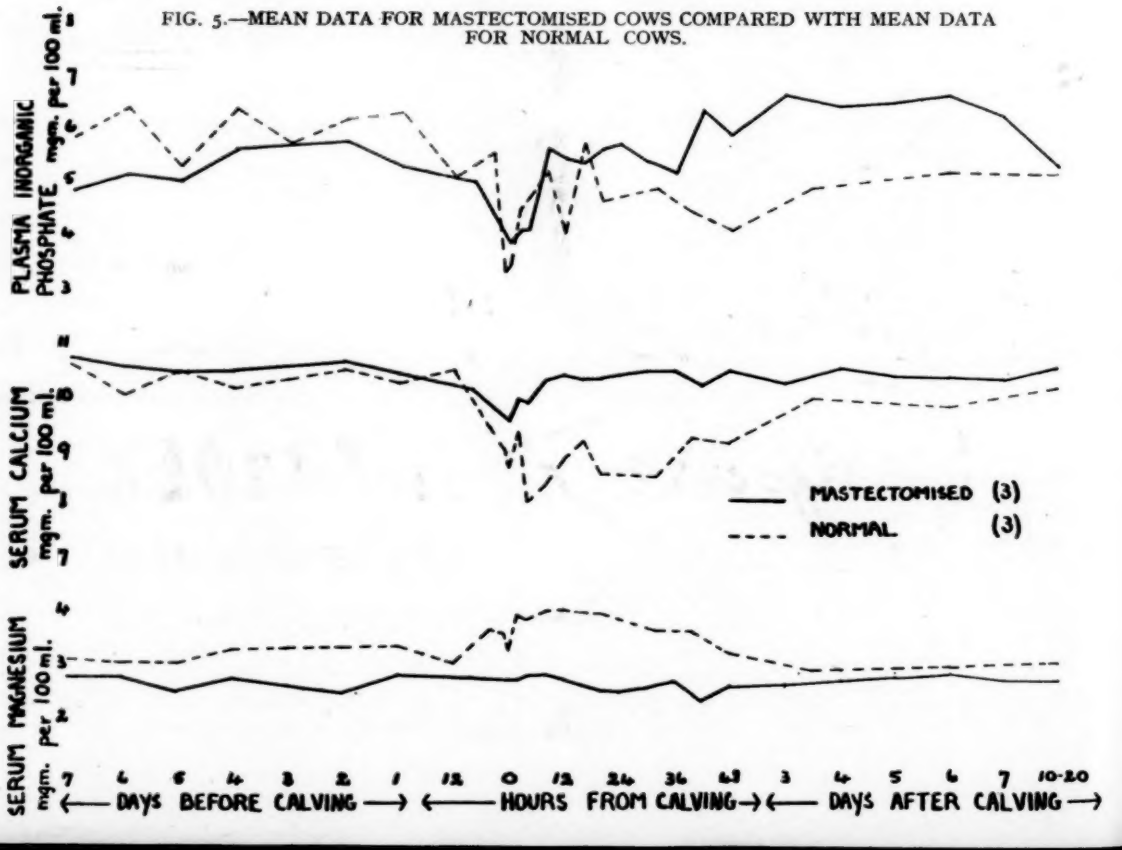
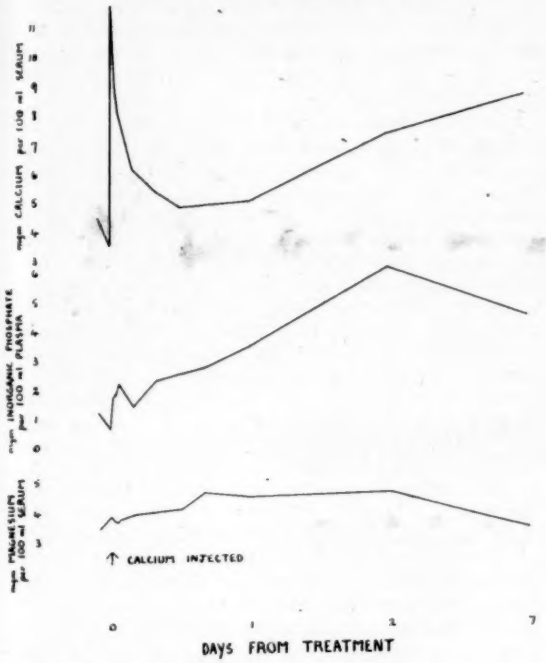


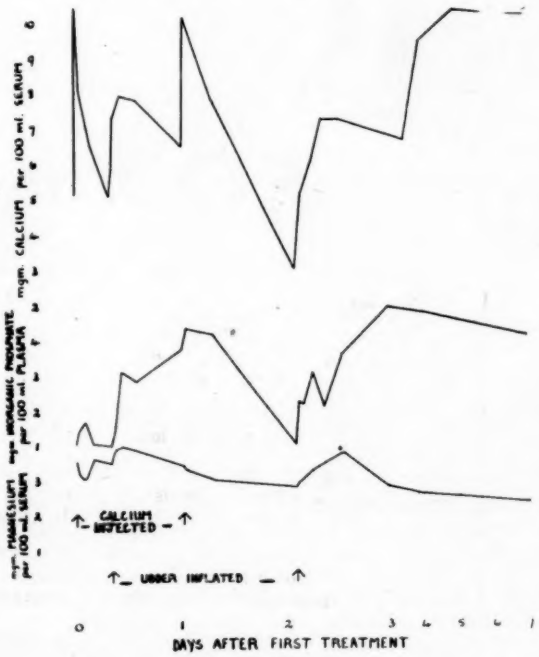
FIG. 6.—MILK FEVER CASE No. 61.


mgm. CALCIUM per 100 ml SERUM
mgm. INORGANIC PHOSPHATE per 100 ml PLASMA
mgm. MAGNESIUM per 100 ml SERUM
↑ CALCIUM INJECTED
DAYS FROM TREATMENT

borogluconate though that can on occasion be quite effective; quite obviously, however, subcutaneous injection would be desirable as well in order to prolong the effect. The other noteworthy feature is the close association of recovery with a marked rise in the inorganic phosphate fraction. It is tempting, of course, to assume from such results that milk fever cannot be considered as a simple hypocalcaemia and that the level of blood inorganic phosphate is of significance in determining the nature of the response to treatment; but we must keep in mind the possibility that this may be an illusion due to the time relationships of satisfactory and unsatisfactory responses. The exact clinical significance of these blood phosphate changes is, I am afraid, still obscure.

All I would like to say in conclusion is that our findings, which I have outlined, confirm that the drain of calcium into the colostrum is the main cause of the fall in blood calcium in normal cows at parturition. If we accept the view that milk fever is an exaggeration of these normal changes it would seem reasonable to assume that this drain of calcium is the major cause of the hypocalcaemia of milk fever. It would seem also that clinical milk fever is the result of the animal being less able than the normal cow to cope with the sudden demands produced by the secretion of colostrum. Since even in the days before any rational treatment was available some 30 per cent. of cases recovered, presumably the regulating mechanism which brings about the gradual return to normal levels in healthy cows, could even in some cases of milk fever do this in time to prevent death from the cellular damage produced by prolonged hypocalcaemia. It would seem likely, therefore, that successful calcium therapy by inducing a temporary rise in the blood calcium level abolishes the clinical signs for long enough to allow the calcium-regulating mechanism time for the mobilisation of calcium from the body reserves and re-establishment of normal levels. The chance of relapses occurring decreases with the passage of time after parturition, presumably partly because the calcium-regulating mechanisms have had more time to act and possibly partly because the calcium-rich colostrum is being replaced by normal milk. My guess is, therefore, that in milk fever the calcium-regulating mechanisms are essentially normal and that the basic cause of the disease is the inability of that normal mechanism to mobilise calcium sufficiently quickly from the osseous tissue to meet the demands—possibly due to a decrease in the availability of the calcium in those reserves. Any such decrease could become more marked with advancing age and thus explain the blood picture described above in the normal animals and also the age incidence of milk fever. I certainly think that a more likely explanation than the parathyroid deficiency theory of Dryerre & Greig or the multiglandular dysfunction described by Seekles, for I have difficulty in visualising a dysfunction of such a temporary nature which is so readily rectified by a treatment essentially replacement in effect. But only time will tell which view is likely to be correct.

FIG. 7.—MILK FEVER CASE No. 44.


mgm. CALCIUM per 100 ml SERUM
mgm. INORGANIC PHOSPHATE per 100 ml PLASMA
mgm. MAGNESIUM per 100 ml SERUM
↑ CALCIUM INJECTED ↑
↑ UDDER INFLATED ↑
DAYS AFTER FIRST TREATMENT

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THE DISCUSSION

A discussion followed the reading of this Paper. The following notes summarise it:—

Mr. R. A. Gilmore said he was rather diffident about opening the discussion. His recollection of this subject went back to the early days of this century when they used oxygen cylinders. This was later replaced by udder inflation and very satisfactory it was. This had now been replaced by a multiplicity of injections and he was not able to differentiate between them. He usually tried one and then the other and when he had finished and the animal had not got up, he started all over again and finally ended by pumping up the udder. He wondered, however, if this was really effective—would another dose of calcium not have worked in such cases? He certainly believed that that would be the case.

He wondered if the speaker had any evidence that milk fever had changed in type, for that was his impression. Years ago he got more cases of the frenzied type which would doubtless now be classified as hypomagnesaemia but he seldom saw that now; on the other hand the deep coma type seemed to be commoner; was that because he was seeing cases earlier nowadays? He wondered also whether udder inflation would make mastitis more prevalent nowadays than it used to; or was it that people were less concerned about mastitis in the past and just fattened the cow without saying anything more about it? Had the speaker any information on the use of Calten AT.10? He had used a dozen doses so far and doubted if it was of any value whatsoever. He would like to know if there was any routine he could adopt to prevent milk fever, *e.g.*, by giving the injections beforehand to a valuable cow which had milk fever in previous lactations. This would be useful on occasions. He had found considerable benefit from the use of strychnine or cocaine in recumbent cases which had been cured of the milk fever but did not get up. Finally, he would like to mention the fact that in the autumn of 1953 they had had a very high mortality in his area and he wondered if that had been reflected in other areas.

Mr. D. McCartney said he would like to know more about the normal calcium and phosphorus levels in cows that had more than four calves; did these alter much after the fourth calving? He had noticed a phenomenon with a recumbent cow which might be of interest, *viz.* that during the calcium injection the animal often stopped breathing for a short period. Would this be due to shock or was there any other explanation?

With regard to Mr. Gilmore's question about change in type of milk fever he wondered if there was any correlation between that and the improved grassland management of recent years. He had one interesting experience in regard to udder inflation that he would like to mention. One cow which had failed to respond to the usual calcium therapy was being milked out preparatory to udder inflation when it rose before the actual inflation took place. Was there any explanation for this?

Mr. G. Hobson, recalling the days when oxygen therapy was used, said that when an animal was treated and had a relapse another inflation with oxygen could be fatal. The same was true if first treated with oxygen and then relapsing it was treated with ordinary air inflation. Was there any likely explanation of this? He would like to know the speaker's views as to the question of breed differences. In his experience Jersey cows were much the most difficult to treat, followed probably by Ayrshires. He would like to know the reason why the old-time treatment of blowing up cured a cow. He also felt that there was some correlation between climate and the incidence of milk fever for he had noticed that the disease was commonest in hot sultry weather. He had also noticed that in cases which occurred long after calving, the animals were usually in oestrus just before the collapse; was there any relationship between these two events? He felt sceptical about the use of Calten, because it was said to increase absorption from the bones and he wondered if that would lead to weakening and possibly encourage rupture of tendons.

Mr. S. G. Stewart asked whether the nervous system was involved in milk fever, and whether the treatment by udder inflation stimulated the nervous system. He thought there might be some significance in the fact that milk fever appeared to be more prominent nowadays when calves were taken away from their mothers at birth, whereas in the old days they were often left to suckle for a few days which he thought might help to reduce the incidence of milk fever.

Mr. G. H. Stewart asked whether there was any evidence that cod liver oil prevented milk fever. He had had one or two cases where this seemed to work.

Mr. D. R. Marshall asked whether any other diseases had any effect on the calcium content of the blood. He had noticed that cows which had taken milk fever on one or two occasions and then got mastitis did not go down at the next calving and he wondered whether the mastitis had any influence on the blood levels. Was there any evidence that a lowered food intake was useful in preventing this disease?

Mr. T. H. Allen asked whether, in those cases which eventually die and are found on *post mortem* to have a fatty liver, this was part of the milk fever syndrome.

Mr. Bolton said that in his experience cows hardly ever ran into the trouble before their fourth calving and seldom after their sixth. Was there any significance in this observation?

Mr. McFerran mentioned two cases which he had in animals at grass, one two days after calving and the other a week after. He wondered whether the grazing had any connection with them.

Mr. Hunniford asked which was the best injection to use—the complex mixture of calcium magnesium and phosphorus or calcium alone? Also which did

the speaker think the better arrangement—to inject these subcutaneously or intravenously?

Dr. W. O. Brown wondered whether the speaker had made any observations on the alkali reserve in his cases.

Mr. H. N. Ferguson said he noticed that in the cases described all the calcium injections had been given intravenously and that involved a sudden rise and sudden drop. Would it not have been better to give them subcutaneously and get a more prolonged rise? He found that if a cow was down and given calcium intravenously you got a good response but you were in danger of getting a relapse; while with a second 4 oz. subcutaneously he seemed to get fewer relapses. In cases at grass he usually found that one injection only would work; but in the autumn once cattle left the grass there were more relapses following one injection. Was it due to the season or was it the method of injection?

Mr. Gordon wondered whether one could judge the approximate levels of other ions beside calcium from the symptoms?

THE REPLY

In reply Professor Robertson said he had to compliment Mr. Gilmore on his opening remarks. He felt that Mr. Gilmore had been unduly diffident. He would not comment on the oxygen cylinder treatment as he had had no experience of it but the remarks Mr. Gilmore had made did illustrate the art of veterinary practice with which Mr. Gilmore was doubtless more familiar than he was. He agreed with Mr. Gilmore that there was probably nothing specially advantageous about udder inflation following a number of relapses for it could quite well be that if the calcium therapy had been used at that stage it would also have been effective. In his experience milk fever had changed in type in his own particular area and he felt that it would vary from time to time and from area to area depending on the association between milk fever and other syndromes such as hypomagnesaemia. It was possibly due in part also to their seeing cases earlier nowadays than 20 or 30 years ago. He felt that udder inflation was just as likely to have caused mastitis in the old days as it was now and possibly more so, but it might well be that people did not worry so much about it 20 or 30 years ago. He had had no experience of the use of AT.10 in this condition and he knew of no effective routine to prevent milk fever although there had been a suggestion recently that large doses of vitamin D just prior to parturition were helpful. He had had no experience of treating recumbent cases with cocaine as mentioned by Mr. Gilmore. In his view there were a number of possible explanations for these recumbent cases—sometimes they might be due to ruptured tendons, sometimes owing to strained muscles, sometimes possibly to pain and stiffness and sometimes possibly due to sheer cussedness! They had had experience in the Edinburgh area occasionally of spells when quite high mortality was evinced in milk fever but not in 1953. He thought again that this would vary from time to time and from area to area.

With regard to Mr. McCartney's question he could confirm that the calcium and phosphorus levels did tend to drop with successive calvings after the fourth but they had too few cases to be of significance and therefore had lumped them all together. The question of the association of the improved grassland management and the milk fever was one that warranted further exploration. It might possibly be associated with increased fertiliser treatment of those pastures.

He agreed with Mr. Hobson that Jersey cows appeared to be more difficult to treat although he had had no particular experience that Ayrshires were worse than many others; he had, however, come across very few cases in Friesian cows. The reason why udder inflation cured a cow seemed to be partly due to the stopping of secretion of milk and partly to the return of calcium to the blood. There appeared to be no correlation between climate and the incidence of milk fever. He did not think the nervous system was primarily involved in milk fever although, of course, a drop in calcium ions in the blood did affect the excitability of nerve endings. There was no reason to think that leaving the calf to suckle for a few days would necessarily reduce the incidence of milk fever; certainly there was evidence to suggest that partial milking had no great effect on incidence.

With regard to Mr. Stewart's query about cod liver oil, as he had mentioned earlier there were claims that this could prevent milk fever when administered in large enough doses just before calving.

He had been asked about the effects of other diseases on the calcium content of the blood; Greig in his original observations in 1930 had quoted 82 cases of cows suffering from other diseases than milk fever in which the calcium figures were within normal limits. There was no evidence that lowered food intake would prevent milk fever any more than there was satisfactory evidence that "steaming-up" increased the incidence.

With regard to Mr. Allen's query about fatty liver he did feel that it was likely that in some of these cases the fatty liver was part of the normal parturition picture combined possibly with a degree of undernutrition and had nothing to do with the milk fever. There were, of course, many different causes of fatty liver and possibly some of the types were pathological.

So far as Mr. Bolton's observations were concerned, they had cases of cows up to their eighth and even later calvings going down with milk fever but, of course, the number of such animals was very small and, therefore, it was not easy to comment on the incidence.

In reply to Mr. Hunniford he would say that there seemed little point in using these combined calcium, magnesium and phosphorus injections except in cases showing definite signs of hypomagnesaemia. Apart from those he thought calcium alone was as effective, and in most cases he would be inclined to give an intravenous injection and a subcutaneous injection to guard against relapses. He had found no evidence in the few studies they had made that the alkali reserve was significantly different in milk fever cows from that in normal cows. Nor had he any evidence of a seasonal incidence in milk fever apart from the

(*Continued at foot of col. 1, overleaf*)

News and Comment

INTERNATIONAL FRIENDSHIP

We continue to receive from veterinary surgeons overseas—notably from France—requests for holiday exchanges between their families and the families of British veterinarians. Our readers have been generous in offering hospitality, but it is hoped that this will be still further increased as more and more requests are being made. We believe there are many who may agree that such contacts between individuals are not in their way less valuable—and are probably more enduring—than contacts publicly made between governments or politicians. At present there are more than 20 enquiries from France alone, and readers are invited to ask for details. Letters should be addressed to the General Secretary of the B.V.A.

A LINK WITH THE ALBERT COLLEGE

The death of Professor L. P. Gamgee (Emeritus Professor of Surgery at the University of Birmingham), which occurred on March 1st, has occasioned the loss of another member of a remarkable scientific family having links with the veterinary profession. Professor Gamgee's father was Mr. Samson Gamgee, inventor of Gamgee tissue, while his grandfather was the famous veterinary scientist and teacher, Professor John Gamgee (1830-1894) who founded the New Veterinary College, Edinburgh in 1859, and whose name will always be linked with the first International Veterinary Congress. The College, according to an article in THE VETERINARY RECORD of December 29th, 1894, having achieved some degree of distinction, was removed to London under the name of the Albert College in 1866; but it was unsuccessful, and was closed two years later. The same article describes Gamgee—the son of a British veterinary surgeon practising in Florence—as one who, with Barlow, introduced scientific method into veterinary teaching for the first time.

Among other famous members of the Gamgee family was a cousin of the late Professor, Sir D'Arcy Thompson, who achieved a world reputation and was for many years Professor of Natural History at St. Andrews.

Continued from previous page.

seasonal incidence of calving which depended, of course, on the farmer's milk production programme.

With regard to Mr. Gordon's question there was the same evidence of a drop in inorganic phosphate as there was of a drop in calcium in relation to the severity of the symptoms.

In conclusion he would like to thank them for the way in which his paper had been received, for the very extensive discussion and for the hospitality given to him that afternoon. He trusted that the observations he had made had been of value to them at least in making their cases more interesting when they realised what sort of blood changes were taking place in association with the clinical symptoms they observed.

ASSOCIATION OF POULTRY PATHOLOGISTS

At a meeting held at the offices of the B.V.A. on March 6th, it was unanimously resolved to form an Association of Poultry Pathologists in Great Britain. The title is intended to include all veterinarians interested in poultry disease problems. A small provisional committee consisting of Dr. R. F. Gordon (Chairman), Dr. W. P. Blount (Secretary, England and Wales), Mr. J. E. Wilson (Secretary, Scotland and Ireland), and Mr. J. D. Blaxland, was appointed to draw up a constitution and to make arrangements for an inaugural meeting. It is hoped that this will be held during Congress week. Some 40 members have decided to support this Association and fuller details will be sent to them in due course. The Committee will be grateful if other interested veterinary surgeons will make direct contact with either of the secretaries.

THE ROYAL COLLEGE OF VETERINARY SURGEONS

It is clear from the acceptances already received that there will be a distinguished and representative assembly at the Annual Dinner of the Royal College to be held at the Hyde Park Hotel, London, S.W.1, on April 12th next.

The Minister of Agriculture, the Rt. Hon. D. Heathcote Amory, M.P., in proposing the toast of the Royal College will have supporting him from his Ministry his Parliamentary Secretary, Mr. G. R. H. Nugent, M.P., as well as the Deputy Secretary, Sir George Dunnett, whilst from the Department of Agriculture for Scotland will come Sir Alexander Glen.

From the Socialist Shadow Cabinet there will be present the Rt. Hon. Tom Williams, M.P., a well-remembered former Minister of Agriculture, who will reply to the toast of The Guests, together with his former Parliamentary Secretary, the Rt. Hon. George Brown, M.P., more recently a Minister in his own right.

The diversity of interest that will be represented is shown by the number of bodies, in addition to the President of the British Veterinary Association, who will be represented by their president or another principal officer. These include the Ministry of Health, the Colonial Office, the Agricultural Research Council, the University Grants Committee, the Pharmaceutical Society, the Association of British Pharmaceutical Industry, the National Farmers' Union, the National Cattle Breeders' Association and many other bodies.

Two prominent names from the agricultural press include Mr. Anthony Hurd, M.P., and Sir Richard Haddon.

Tickets for the dinner at two guineas each are still obtainable from the Registrar, 9-10 Red Lion Square, London, W.C.1.

THE NORTH WALES DIVISION

Members of this Division are asked to note that a meeting will be held in the Council Chamber, Town Hall, Ruthin, on April 6th, at 2 p.m. Mr. G. Wyse, the senior Vice-President, will be in the chair, and routine business will be followed by an address from Professor H. D. Kay, Director of the National Institute for Research in Dairying on the subject "Recent Aspects of the Problem of Non-Fatty Solids in Milk."

PERSONAL

Births

BOWDITCH.—On March 10th, 1956, to Sheila (*née* Scott) and Edmund Bowditch, M.R.C.V.S., of 29, William Street, Windsor, a son, brother for Robert and Matthew.

FULLER.—On February 28th, 1956, to Anne (*née* Knowles) wife of A. T. Fuller, 22, London Road, Sittingbourne, a son (John Barry) brother for Caroline.

HEDGER.—On March 9th, 1956, at Eldoret European Hospital, Kenya Colony, to Sylvia, wife of R. S. Hedger, M.R.C.V.S., a son, brother for André.

LINDLEY.—On March 16th, 1956, at Jos, Nigeria, to Doreen (*née* Eggleton) wife of Philip Lindley, a brother for Roger—Robert Howard.

POLLEY.—On March 23rd, 1956, to Kathleen, wife of Maurice J. Polley, M.R.C.V.S., 45, Pack Lane, Basingstoke, a son, Simon Nicholas.

SPALDING.—On February 9th, 1956, at 95, Damers Road, Dorchester, Dorset, to Nancy, wife of V. T. Spalding, M.R.C.V.S., a daughter, Mary-Anne Beatrice Eileen.

Marriages

RICHARDSON—SHEEHAN.—At Spencer St. Church of the Annunciation in Chesterfield, on January 14th, 1956, Robert Richardson, B.SC., M.R.C.V.S., 81, Newbold Road, Chesterfield, and Betty Sheehan, "Airmount," Enniscorthy, Co. Wexford, Eire.

SIMPSON—DUNN.—On March 15th, 1956, in London, Jean, daughter of Mr. and Mrs. Scott Dunn of Benfield House, Hangleton, Hove, to Dr. Keith Simpson, Weymouth Street, London.

R.C.V.S. OBITUARY

We record with regret the deaths of the following members of the profession:—

ANDERSON, Robert Gray, O.B.E., T.D., F.R.C.V.S., 5, Whytehouse Avenue, Kirkcaldy, Fife, Major late R.A.V.C. (T.A.). Graduated December 18th, 1897, Glasgow; Fellow December 7th, 1912. Died March 18th, 1956, aged 80 years.

JELBART, George Henry, 59, Manor Road, Brackley, Northants. Graduated May 24th, 1906, Edinburgh. Died February 27th, 1956, aged 77 years.

COMING EVENTS

April

4th, 5th and 6th (Wed., Thurs. and Fri.). B.V.A. Council and Committee Meetings at the Royal (Dick) School of Veterinary Studies, Edinburgh.

5th (Thurs.). Annual General Meeting of the Lakeland (proposed) Division, B.V.A., at the Hydro, Windermere. A dinner will follow the meeting.

6th (Fri.). Meeting of the North Wales Division, B.V.A., Ruthin, 2 p.m.

7th (Sat.). 10th Annual Dinner of the B.R.X. Club at the Royal Veterinary College, 6 p.m.

11th and 12th (Wed. and Thurs.). L.A.B. Congress at Royal Veterinary College, London.

12th (Thurs.). Annual Dinner of R.C.V.S. at Hyde Park Hotel. Tickets two guineas each obtainable from the Registrar.

13th (Fri.). Symposium on "Infections in Laboratory Animals" by the Laboratory Animals Bureau at the Royal Veterinary College, London, 9.45 a.m.

18th (Wed.). Meeting of the Lancashire Division, B.V.A., at the University of Liverpool Veterinary Field Station, Neston, Wirral, 12 noon.

19th (Thurs.). Ninth Ordinary Meeting of the Southern Region of the A.V.T. and R.W's. at the Royal Veterinary College, London, N.W.1.

20th (Fri.). Meeting of the Mid-West Division, B.V.A., at the Berkeley Café, Clifton, at 2.30 p.m.

26th (Thurs.). Meeting of the Essex Division, B.V.A., in the Essex Institute of Agriculture, Writtle, Essex, at 7.30 p.m.

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

Our readers will understand that a number of letters were held over during the long period of the printing dispute. We publish two of them below.

Wounds in the Teats of Cows

Sir,—From time to time we read of methods of suturing devised to repair injured teats in the lactating cow, particularly in cases where the milk duct has been laid open. We cannot remember, however, that anyone has ever stressed the fact that the bovine teat shows little tendency to healing by first intention, and in our experience is even slow to granulate. Someone may say the reason for this failure to heal by first intention is due to want of effective closure of the wounded duct; this, however, is not the explanation. In our experience, wounds of the teat, without any injury to the teat duct, show just as little tendency to heal by first intention. Again, it may be argued that a wounded teat is difficult to heal because of the abuse to which it is bound to be subjected as a result of being milked twice a day.

Recently we conducted an experiment on two teats encountered on consecutive days, both of which had wounds penetrating the milk ducts.

The first was a Guernsey cow which had gone down with milk-fever within 24 hours of calving. In blundering about she had apparently trodden on a hind teat and cut it, not far from the sphincter, opening the duct. We went to some trouble to close the duct using "twenty-day gut" sutures, close together, and then closed the outer wound with one

Halstead and several nylon interrupted sutures. We next inserted a plastic tube, the lower end of which is fitted with a screw cap. This is an American product, the idea of which is to facilitate the milking of injured teats. It seems to be quite effective, and is operated simply by unscrewing the cap at the bottom of the tube when the milking-cups are applied to the three sound quarters. In this case the herdsman was able to get half a gallon of milk daily from the injured teat. The sutures were removed on the ninth day and, though prior to removal the wound looked as though it might heal, it just fell apart as though it had never been sutured.

The other case was that of a second-calved Fresian cow due to calve in two months, in process of being "dried off." She had wounded a hind teat, the wound extending into the milk-duct with milk escaping. The external wound in this case was not likely to heal easily, as quite an area of the teat had been skinned and the owner was warned that however it was sutured, the skin was likely to slough. We cast the animal and sutured the wound as we had done in the case previously described. As we had not another tube adapted for the purpose we inserted a piece of Polythene drainage tube, kept in place by a nylon suture. Although there was a perpetual drip of milk from the tube in this case (scarcely perceptible at times) when the other three quarters were being milked quite a flow of milk came from the injured teat. Exactly one week later the skin in the outer wound sloughed, the quarter became affected with mastitis, and milk began to come through the wound.

Lest it be thought that our technique was inadequate let us recount what happened in the case of a valuable cow in which we were asked to deal some years ago with a milk fistula. We made our attempt just as the animal was "dried off." We need not describe the operation, suffice it to say that when the sutures were removed on the ninth day the whole wound opened up. Thinking that the value of the cow warranted the expense we enlisted the services of an eminent surgeon, who, if he did not say so, smiled so indulgently as to leave us in no doubt that he considered us country cousins in matters of surgery. Our instructions were to remove the stitches in 10 days; this we did only to find that everything fell apart in the same disconcerting fashion as before.

No doubt the reason for all these failures, and many more besides, has to do with the histology of teat tissue. Even if one had never heard of histology, contemplation of the ability of the teat to stretch and bend in all directions without apparently suffering any injury might suggest that paucity of blood vessels is one reason.

It has occurred to us that MacClean's knife might not be such an effective instrument as it is, were the aptitude to granulate in teat tissue as marked as, for example, that of the udder wall itself. This has seemed to us nothing short of miraculous at times. However, we are more concerned with finding some effective way to expedite granulation in teat surgery. Can some form of mild irritant or stimulant be devised for application to these wounds?

We should like to hear the views of other practitioners on the subject; and if some bright young surgical research worker is in need of a problem, may we commend this one to him as a job very well worth while?

Yours faithfully,

HORACE BELL.

67 Grenfell Road,
Maidenhead,
Berks.

February 20th, 1956.

Treatment for the Retention of the Afterbirth in Cattle

Sir,—For over 30 years, after the removal of the afterbirth, I have made it a rule to douche the uterus with a reliable antiseptic: before we had the modern ones I used salt. In the old days we had only lysol and the common coal tar products; chinosol was sometimes used, but it was not as reliable as salt. I may mention, for the interest of junior members of the profession, that we carried round with us 1 lb. packets of common salt, and used half a packet to each bucket of water. Never do I remember any ill effects, and certainly one had few cases of leucorrhoea. Before retiring from practice I can scarcely remember an example of this condition, yet last year, when I was assisting a member in his practice, cases were of daily occurrence. I never saw more than during that week, but I was not sufficiently long in the practice to know the cause.

In my old practice I never used stilboestrol, but last summer I acted as locum tenens in the Midlands where the veterinary surgeon never cleansed a cow. His treatment was 3 c.c. of stilboestrol followed on the second and fourth day with 1 lb. mag. sulphate. He told me he had no intention of returning to the old method of removing the afterbirth. While I was there I naturally did as he did, and I should like to have followed cases up had I been there for a longer period.

Mr. Mayor uses the pessaries he describes in his paper (*Vet. Rec.* **68,** 111) on the third and fourth days after calving. He says the membranes are separated with ease after waiting a further two days. That is only what one would expect. It is never advisable to cleanse a cow before the third day for a full-time calf, and four days should be allowed for abortion cases. If that principle is observed most afterbirths will come away without much trouble. If the membranes are still not ready for removal give a good douche with disinfectant, using one or two buckets of water. When you return after two days there will be either no cleansing needed, or very little, and no leucorrhoea need be anticipated.

Yours faithfully,

G. C. LANCASTER.

Eastwick House,
Evesham,
Worcs.

February 11th, 1956.